initiation of the immune response seems to take place. These findings from a murine GN model suggest that the transfer of in vitro expanded autologous Treg might represent a new treatment option in human GN.

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Aldosterone-mediated endothelial remodeling and oxidative stress

To the Editor: The recognition of the inflammatory and profibrotic role of aldosterone in the pathophysiology of cardiovascular disease, via its effect on endothelial dysfunction, is of growing importance, as demonstrated by the results of the recently concluded Randomized Aldactone Evaluation Study (RALES) [1], and the EPlerenone neuroHormonal Efficacy and SUrvival Study (EPHESUS) [2]. These studies have, in fact, indicated the reduction of aldosterone effects through receptor blocking as additional benefit to patients with cardiovascular diseases.

In a paper published in the May issue *Kidney International*, Oberleithner documented "in vitro" an aldosterone remodeling effect on human endothelium through induction of cell stiffness due to a presumably aldosterone induced oxidative stress via modulation of NAD(P)H oxidase [3]. We would like to provide further support to the contention of a specific remodeling and profibrotic action of aldosterone with the demonstration "ex vivo" in human mononuclear cells, recently published by our laboratory [4], that indicates that aldosterone has a direct effect on oxidative stress through its ability to increase the levels of $p22^{phox}$, an important subunit of NADPH oxidase, essential for superoxide anion generation. It, in fact, functions as an integral subunit of the final electron transport from NADPH to heme and molecular oxygen in generating superoxide anions. The aldosterone-induced increased level of PAI-1, a recognized profibrotic protein, we have shown in the same study [4], may also provide a direct link to the cardiovascular profibrotic and remodeling action of aldosterone. Our findings were further strengthened by similar effects shown by glycyrrhetinic acid [4], a constituent of licorice root, which is known to have a direct mineralocorticoid-like effect [5]. Thus, the report of Oberleithner [3] in combination with the results of our study provides clear evidence for the aldosterone-related vascular remodeling effects through its induction of oxidative stress and oxidative stress-related profibrotic molecules, such as PAI-1 [4].

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Screening for microalbuminuria

In their recent contributions to *Kidney International*, de Zeeuw [1], as well as de Jong and Brenner [2], argue that primary prevention of cardiovascular and renal disease may be possible by lowering albumin excretion in